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PASSIVE SMOKING AND LUNG CANCER IN SWEDISH WOMEN

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The relation between passive smoking and lung cancer was examined by means of a case-control study in a cohort of 27,409 nonsmoking Swedish women identified from questionnaires mailed in 1961 and 1963. A total of 77 cases of primary carcinoma of the bronchus or lung were found in a follow-up of the cohort through 1980. A new questionnaire in 1984 provided information on smoking by study subjects and their spouses as well as on potential confounding factors. The study revealed a relative risk of 3.3, constituting a statistically significant increase ($p < 0.05$) for squamous cell and small cell carcinomas in women married to smokers and a positive dose-response relation. No consistent effect could be seen for other histologic types, indicating that passive smoking is related primarily to those forms of lung cancer which show the highest relative risks in smokers.

histology; lung neoplasms; smoking; tobacco smoke pollution

In recent years there has been a growing interest in the health effects of environmental tobacco smoke. Biologic monitoring has demonstrated that exposure to tobacco smoke constituents may be appreciable among passive smokers (1–4). Several studies show that children with parents who smoke have an increased risk of bronchitis and pneumonia, and some data also indicate changes of pulmonary function in adults and children exposed to environmental tobacco smoke (5).

A few epidemiologic studies have been published on passive smoking and lung cancer (6–17). Some of these show increased risks for nonsmokers married to smokers, but the results are not fully consistent. Most of the studies were not specifically designed to investigate effects of passive smoking, and there are various potential sources of random and systematic errors which make it difficult to interpret the findings. One aim of the present investigation was to try to minimize such errors, especially with regard to the validity of the information on exposure and effects.

MATERIALS AND METHODS

Study subjects

This investigation is designed as a case-control study within a cohort of nonsmoking women. There are two sources for the cohort. Most of the subjects are taken from a sample of about 55,000 men and women aged 15–65 years in the 1960 National Census of Sweden for whom tobacco smoking was investigated by a questionnaire mailed in 1963. Detailed descriptions of the sampling strategy and the questionnaire are

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given elsewhere (18). The response rate among the women was 95.4 per cent. A total of 17,679 (66.8 per cent) of the women stated that they had never smoked any form of tobacco, and these are included in the present study.

The second source of subjects is the "old" Swedish twin register which contains about 11,000 same-sex twin pairs born between 1886 and 1925 (19). The twins were identified from birth certificates, and a questionnaire was mailed to them in 1961, primarily to determine zygosity and tobacco smoking status. The response rate among the eligible female twin pairs was 85.1 per cent. In all, 9,730 women (80.6 per cent) had never smoked, and they make up the rest of the study cohort.

Cancer morbidity and mortality of the 27,409 women in the study cohort were determined through 1980 in the Swedish Cancer Register and the National Register on Causes of Death, respectively. The quality of the information in these registers is high for most cancer diagnoses (20). A total of 92 cases of tracheal, bronchial, lung, or pleural cancer were identified (*International Classification of Diseases* (ICD), Seventh Revision, codes 162–163) (21). These subjects constitute the case series.

Two control groups, each containing two controls per case, were also selected from the study cohort. Control group 1 consisted of subjects who were matched to their respective case on year of birth (±1 year). Control group 2 included subjects who were matched on year of birth as well as on vital status at end of follow-up. The subjects in both control groups were selected at random from subjects who fulfilled the matching criteria, with the exception that no woman could be used as a control for her twin sister. The entire study group consisted of 460 subjects: 58 cases and 232 controls from the 1963 smoking sample, as well as 34 cases and 136 controls from the twin register.

Exposure information

There were two sources of exposure information. First, as described above, data in the 1961 and 1963 questionnaires were used to define the cohort from which the cases and controls originated. The second source was a questionnaire mailed in 1984 to each study subject or, if she was dead, to the next-of-kin (excluding the husband), in order to validate the data on smoking as well as to assess the exposure to environmental tobacco smoke from husbands and parents. If a woman had been married more than once, smoking was investigated only for the man with whom she had cohabited the longest. Questions on occupational and residential history were also included. If the questionnaire answers were incomplete, additional information was obtained by telephone interview. The methodology using next-of-kin to obtain data has been shown to provide exposure information of high quality (22–24).

The residential history information from the 1984 questionnaire included data on addresses (parishes) and types of houses in which the study subjects had lived. A parish was classified as urban if 90 per cent or more of the population lived in built-up areas according to the 1970 National Census. One-family houses made of material other than wood and with basements were classified as dwellings presenting a greater risk of radon exposure. Indoor radon measurements show that the average concentrations in such houses are higher than in other common types of dwellings in Sweden (25).

Statistical methods

Several methods have been used in the statistical analysis. The matching was retained in some analyses, and maximum likelihood estimates of relative risks (approximated with odds ratios) and exact confidence intervals were computed according to the method of Miettinen (26). In other analyses, the matching was dissolved, and the relative risks and confidence intervals were estimated as suggested by Mantel and Haenszel (27) and Cornfield (28), respectively. The method proposed by Mantel (29) was used to test linear trends in these analyses. Besides the conventional strati-

fied analyses, a conditional logistic regression analysis (30) was carried out in an attempt to control residual confounding in the risk estimates and to study interactions.

RESULTS

A careful review of the medical records of the 92 lung cancer cases showed that in nine cases the primary site was not the bronchus or lung (there were no primary tracheal or pleural carcinomas), and in six cases the primary site was uncertain. Carcinoma of the breast, which occurred in five cases, was the most common cause of secondary carcinomas. For 64 of the 77 primary carcinomas of the bronchus or lung, the diagnoses were based on histologic evidence, and for 12 diagnosis was based on cytology. In one case, an autopsy was performed, but there was no histologic examination.

The distribution of histologic types among the primary bronchial and lung carcinomas is shown in table 1. The classification is based on the information in the medical records, particularly the pathology reports. Adenocarcinoma is the most common group, constituting 57.1 per cent of the total. Squamous cell and small cell carcinomas constitute 31.2 per cent. The average ages at diagnosis and at death for the whole group of carcinomas are 69.0 and 69.6 years, respectively. In the following analysis, the squamous cell and small cell carcinomas are grouped together because these types have generally shown the highest relative risks among smokers (31).

TABLE 1

Histopathology of primary bronchial and lung carcinomas and mean ages at diagnosis and at death in a cohort of 27,409 nonsmoking Swedish women

Diagnosis	No.	%	Age (years)	
			Diagnosis	Death
Squamous cell carcinoma	12	15.6	68.5	70.1
Small cell carcinoma	12	15.6	65.6	65.8
Adenocarcinoma	44	57.1	69.7	70.2
Large cell carcinoma	5	6.5	67.9	68.0
Other primary carcinomas	4	5.2	74.4	74.8
Total	77	100.0	69.0	69.6

Table 2 shows the distribution of selected variables among the cases and the control groups. As a result of the matching criteria, the age distribution and vital status are similar for the cases and control group 2. In control group 1, there is a shift toward older ages, and more subjects were alive at the end of follow-up than in the two other groups.

Questionnaires were returned for 90.2–96.7 per cent of the study subjects in the different groups. Among the proxy respondents, 68.4 per cent were children of the study subjects, 21.3 per cent were brothers or sisters, and 10.3 per cent were other relatives. There were no differences in the type of proxy respondents between the case and control groups.

All of the returned questionnaires contained information on smoking by the study subject and, with the exception of one subject in each control group, on whether she had been married and whether her husband had smoked. For the other questionnaire items, e.g., smoking habits of parents, employment, and residential history, the internal nonresponse rates ranged from 9.6–32.6 per cent. The percentages in table 2 are based on the number of respondents to each item.

Eight (1.8 per cent) of the 436 women for whom questionnaire information could be obtained in 1984 had smoked daily during at least two years. Four of these had stopped before answering the 1961 or the 1963 questionnaire, and one had started after that. Two women smoked 1–7 cigarettes per day, and one was a pipe smoker. These eight women were excluded in the subsequent analyses. There were no pronounced differences between the groups with regard to the percentage of women who were married or the percentage who were married to smokers.

For the remainder of the questionnaire items, no consistent differences were seen between the groups, with the possible exception of a tendency toward a larger per-

TABLE 2

Distribution of selected variables among cases of lung cancer and two control groups matched for year of birth, from a cohort of nonsmoking women

	No.			%		
	Cases	Control group 1	Control group 2	Cases	Control group 1	Control group 2
Total	92	184	184	100	100	100
Localization of primary tumor						
Bronchus or lung	77			83.7		
Other site or uncertain	15			16.3		
Age at death or at end of follow-up (years)						
40–69	44	38	93	47.8	20.7	50.5
70–79	40	90	73	43.5	48.9	39.7
80–91	8	56	18	8.7	30.4	9.8
Vital status at end of follow-up						
Alive	5	121	10	5.4	65.8	5.4
Dead	87	63	174	94.6	34.2	94.6
Total questionnaire respondents	83	178	175	90.2	96.7	95.1
Smoked daily†	2	3	3	(2.4)*	(1.7)	(1.7)
Married†	70	143	151	(84.3)	(80.3)	(86.8)
Married to smoker†	37	76	77	(44.6)	(42.9)	(44.3)
At least one parent smoker†	12	30	21	(21.1)	(21.4)	(15.9)
Employed outside home†	33	73	52	(44.0)	(48.3)	(34.7)
Lived in urban area†	39	78	82	(60.9)	(61.4)‡	(62.6)
Lived in dwelling presenting a greater risk of radon exposure†	11	13	9	(17.2)	(10.8)‡	(7.0)

* Numbers in parentheses correspond to percentages of total number of questionnaire respondents to each item.

† Minimum duration of two years.

‡ Exposures occurring after the death of their respective case have been excluded for controls alive at the end of follow-up.

centage of cases than of controls who lived in dwellings presenting a greater risk of radon exposure. A detailed analysis of the occupations held by the cases and controls did not reveal any differences between the groups. The great majority of the occupations were in the service sector and typical for women of the age group under study, e.g., housemaid, cook, seamstress, cleaner, and nurse.

In the following analyses, the 15 cases with primary sites other than the bronchus or lung have been excluded. Table 3 gives, in a matched analysis, the relative risks for primary carcinoma of the bronchus or lung in women married to smokers. Never married women and women married to nonsmokers constitute the reference category. The results are consistent for both control groups. Pooling the control groups produces a relative risk of 3.3 for squamous cell and small cell carcinomas (95 per cent confidence interval (CI) = 1.1–11.4) associated with marriage to a smoker. Within this group, the relative risks were increased for both histologic types. The relative risks for the other histologic types and for the entire group are 0.8 (95 per cent CI = 0.4–1.5) and 1.2 (95 per cent CI = 0.7–2.1), respectively.

Table 4 gives a dose-response analysis with regard to smoking by the husband. The matching was dissolved in this analysis as well as in table 5. There is a positive trend in the relative risk for squamous cell and small cell carcinomas ($\chi^2 = 3.9$), but not for the other histologic types. The relative risk in the highest exposure group, i.e., women with husbands who smoked more than 15 cigarettes per day or one pack

TABLE 3

*Relative risks (RR) and 95% confidence intervals (CI) for primary carcinoma of the bronchus or lung in nonsmoking women married to smokers with two control groups in a matched analysis**

Histologic type	No. of cases	Control group 1†		Control group 2‡		Both control groups	
		RR	CI	RR	CI	RR	CI
Squamous cell or small cell carcinoma	20	3.8	1.1–16.9	3.4	0.8–20.1	3.3	1.1–11.4
Other types	47	0.7	0.3–1.6	0.8	0.4–1.7	0.8	0.4–1.5
Total	67	1.2	0.6–2.2	1.1	0.6–2.1	1.2	0.7–2.1

* Never married women and women married to nonsmokers constitute reference category. Maximum likelihood estimates of relative risks and exact confidence intervals (26).

† Matched to cases on year of birth.

‡ Matched to cases on year of birth as well as on vital status at end of follow-up.

TABLE 4

*Relative risks (RR) and 95% confidence intervals (CI) for primary carcinoma of the bronchus or lung in nonsmoking women in relation to estimated exposure to tobacco smoke from the husband**

Histologic type	Never married or married to a nonsmoker		Low exposure to tobacco smoke of husband†			High exposure to tobacco smoke of husband‡			Chi-square for trend§
	No. of cases	RR	No. of cases	RR	CI	No. of cases	RR	CI	
Squamous cell or small cell carcinoma	7	1.0	10	1.8	0.6–5.3	3	6.4	1.1–34.7	3.90
Other types	27	1.0	16	0.8	0.4–1.6	4	2.4	0.6–8.7	0.03
Total	34	1.0	26	1.0	0.6–1.8	7	3.2	1.0–9.5	1.45

* Age-standardized relative risk estimates (27) and approximate confidence intervals (28).

† Husband smoking up to 15 cigarettes per day or one pack (50 g) of pipe tobacco per week or any amount during less than 30 years of marriage.

‡ Husband smoking more than 15 cigarettes per day or one pack of pipe tobacco per week during 30 years of marriage or more.

§ Test for linear trend (29).

of pipe tobacco per week during 30 years of marriage or more, is 3.2 (95 per cent CI = 1.0–9.5) for all histologic types combined.

Table 5 shows the influence of parental smoking on the risk of primary carcinoma of the bronchus or lung, controlling for smoking by the husband. There is no consistent evidence of an effect, and the 95 per cent confidence intervals for the relative risks in women with at least one smoking parent encompass 1.0 for both histologic groups. These results must be interpreted with caution in view of the lack of information on parental smoking habits for 24 per cent of the questionnaire respondents.

The results of the conditional logistic regression analysis, which included cases and matched controls with information on all variables, were consistent with the results of the stratified analyses. There was no important confounding of the association between smoking by the husband and squamous cell and small cell carcinomas by occupation, by living in houses with a greater risk of radon exposure, or by living in urban areas. None of the relative risks associated with these factors deviated significantly from 1.0 upon statistical testing. For all histologic types taken together, the relative risks and 95 per cent confidence intervals associated with marriage to a smoker and with living in a house presenting a greater risk of radon exposure were 1.2 (95 per cent CI = 0.6–2.6) and 1.4 (95

TABLE 5

*Relative risks (RR) and 95% confidence intervals (CI) for primary carcinoma of the bronchus or lung in nonsmoking women in relation to smoking habits of parents**

Histologic type	Both parents nonsmokers		At least one smoking parent		
	No. of cases	RR	No. of cases	RR	CI
Squamous cell or small cell carcinoma	10	1.0	6	1.9	0.5–6.2
Other types	28	1.0	3	0.5	0.1–1.9
Total	38	1.0	9	1.0	0.4–2.3

* Mantel-Haenszel estimates of relative risks (27) standardized for age and smoking of husband with approximate confidence intervals (28).

per cent CI = 0.4–5.4), respectively. For women who had been married to a smoker and who had lived in a house presenting a greater risk of radon exposure the relative risk was 2.5 (95 per cent CI = 0.8–8.5), suggesting a positive interaction between the two variables.

DISCUSSION

The results of our study indicate that exposure to environmental tobacco smoke is related to an increased risk of those histologic types of lung cancer which show the highest relative risks in smokers. This is in general agreement with the findings of Trichopoulos et al. (7), Garfinkel et al. (15), and Koo et al. (16), although these authors looked at somewhat different carcinoma types and/or used other definitions of exposure. It would be of interest to see an analysis of the risks for different histologic types in the other published studies on passive smoking and lung cancer, especially those with an appreciable number of cases, as well as in subsequent studies on this topic.

Combining the published epidemiologic studies provides a weighted average relative risk of lung cancer of 1.5 associated with marriage to a smoker (5). The results of the present study are consistent with this estimate. A 50 per cent increase in risk does not seem unreasonable in view of exposure estimates among passive smokers (5, 32) and the excess risks of between 100 and 900 per cent for smokers in the lowest exposure category, as a rule 1–9 cigarettes per day, in the major cohorts studied (18, 33–39). It should be noted that relative risks for squamous cell and small cell carcinomas would be expected to be even higher, i.e., if the case group is not "diluted" with adenocarcinomas or other types with weaker association to smoking.

Several sources of random and systematic errors have to be considered in the interpretation of the findings. In contrast to earlier studies on passive smoking and lung cancer, the present study has a "double check" on the smoking status of all study subjects. Data were obtained from the 1961 and 1963 questionnaires that were used to define the cohort as well as from the 1984 questionnaire. Our results indicate that misclassification of nonsmokers was a minor problem and that failure to take this problem into account would not severely bias the association between passive smoking and lung cancer. This is supported by the findings of other Swedish studies, which show a high quality of questionnaire information on smoking, both when the data were obtained from the subjects themselves and when data were obtained from next-of-kin (22, 23).

Using smoking by the husband as the only measure of exposure to environmental tobacco smoke will result in misclassifications in the exposure assessment. To the extent that such misclassifications are unrelated to the disease in question, this would tend to reduce any true association between passive smoking and lung cancer. The similar percentages of exposed persons among the cases, excluding squamous cell and small cell carcinomas, and the two control groups suggest that errors in the reporting did not affect the cases and controls differently. This lends further support to the association with smoking of the husbands, which was noted for squamous cell and small cell carcinomas only. Obviously,

it is unlikely that the next-of-kin respondents were aware of the histologic subtypes diagnosed for the cases.

Our results show that poor quality of the diagnosis may be a problem in studies of lung cancer in female nonsmokers. Secondary pulmonary carcinomas or carcinomas with unknown primary sites appeared in about one-sixth of the cases reported in the cancer and/or cause of death registers. This is in close agreement with the findings of Garfinkel (8), which were based on death certificate diagnoses in the United States. If secondary tumors are not excluded from the case series, the relative risks associated with any factor that causes primarily lung carcinomas are likely to be underestimated. As noted previously, the analysis may be further strengthened by separating different histologic types.

Besides the quality of the exposure and diagnostic information, the validity of our study is also affected by the control of confounding factors. The association between passive smoking and lung cancer of the squamous cell and small cell types was not confounded by occupation, urbanization, or living in houses with a greater risk of radon exposure nor were any of these factors associated with a clear increase in risk when passive smoking was controlled. These findings should be interpreted with some caution in view of the internal nonresponse on the questionnaire for items other than smoking of the study subjects and their spouses. It is, however, improbable that uncontrolled confounding by the factors under study explains relative risks of the magnitude observed, as well as the positive dose-response relations. No information was obtained on intake of food items that may affect the lung cancer risk.

Analysis of all the lung cancer cases suggested a positive interaction between marriage to a smoker and living in dwellings presenting a greater risk of radon exposure, i.e., one-family houses made of material other than wood and with a basement. Increased risks of lung cancer associated with living in such houses have been observed previously (40–42), but our study also provides data on exposure to environmental tobacco smoke. Our findings are consistent with an interaction between tobacco smoke and radon daughters similar to the one observed in uranium miners (43) and in smokers living in dwellings with a greater risk of radon exposure (41). It is also of interest to note that the radon daughter concentration has been shown to increase considerably as a result of attachment to aerosol particles in rooms filled with tobacco smoke (44).

In conclusion, our results indicate that exposure to environmental tobacco smoke is related primarily to those forms of lung cancer which show the highest relative risks in smokers. The results are internally consistent and in general agreement with other studies. Our findings are of scientific interest and have public health implications, although it is obvious that lung cancer in passive smokers is a rare phenomenon. The accumulating evidence in children and adults shows that serious health effects can probably result from heavy exposure to environmental tobacco smoke. This should encourage further research, including both exposure assessments and etiologic studies.

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